Exercise. Eat. Repeat. Focus on “Prior exercise training blunts short-term high-fat diet-induced weight gain”

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THE DELETERIOUS EFFECTS of a saturated fat-rich diet and sedentary behaviors on metabolic health are well established. For many years, exercise training has been recognized as a means to treat or attenuate metabolic disease. For example, exercise training is often employed as a weight loss modality in conjunction with and independent of dietary restrictions, aimed at increasing total energy expenditure (6, 11). As a means to improve metabolic function following the onset of insulin resistance or Type 2 diabetes, exercise has proven an effective intervention in animal models (3, 8) and humans (4, 10). Further, there is an increased effort to identify the mechanisms induced by exercise training that allows for treatment of metabolic diseases. However, few studies have focused on the ability of exercise training to prevent insults induced by dietary stressors causing such conditions such as obesity or insulin resistance.

Snook et al. (13) employed a novel study design to investigate whether prior exercise training would diminish weight gain and the disruption of glucose homeostasis following a short-term high-fat diet (HFD). Mice underwent 4 wk of treadmill exercise while consuming a low-fat diet (LFD) before completing a 4-day HFD in the absence of exercise. The authors demonstrated that a 4-day HFD induces weight gain and impairs glucose tolerance in sedentary mice, but that prior exercise training was sufficient to attenuate increases in body weight and glucose intolerance. Snook et al. (13) elaborated upon these findings, demonstrating an increased total energy expenditure (TEE) relative to trained LFD controls that occurred independent of changes in brown adipose tissue mitochondrial function. Collectively, the data suggest that lean body mass appears to be critical in the protection against a short-term HFD, resulting from prior exercise training.

The findings of Snook et al. (13) are not only unique, but efficacious in the human context. Recently, it was shown that maladaptations to an isocaloric HFD occur in as little as 5 days in sedentary humans (1). Whether such early-stage adaptations to a short-term HFD mark the descent toward obesity and insulin resistance is unclear. Previous studies in humans have shown that acute bouts of exercise prior to a high-fat meal reduces postprandial lipemia (5, 7), while supporting mechanisms of vascular endothelial maintenance and repair through the cell-specific elimination of oxidative stress (7). Collectively, these studies highlight that while humans are susceptible to potentially negative metabolic adaptations following only short-term HFDs, even a single prior exercise bout may be capable of defending against the metabolic stress of a high-fat meal. The prospect that the findings of Snook et al. (13) may not only be desirable, but replicable in humans, is an intriguing one that shifts the focus of exercise away from treatment toward one of prevention.

The finding that 4 wk of exercise training may be sufficient to attenuate the negative metabolic outcomes of a short-term HFD is important in the context of advancing our understanding in combating metabolic disease. However, several considerations and questions remain. First, the greater TEE in the trained HFD group was only apparent when comparing the change in TEE between LFD- and HFD-trained mice. No significant differences were present between either of the trained groups and their respective control groups. This may suggest that any increase in TEE following a training period is dependent upon a metabolic insult; thus, although the study design utilized may yield positive weight control benefits when challenged with a positive energy balance environment, it may not be effective under energy-neutral or -negative environments.

Consumption of a HFD increased glucose intolerance, although this effect was attenuated in the trained-HFD group relative to sedentary-HFD group, while glucose tolerance remained unchanged between sedentary and trained mice kept on a LFD. Combined, these data suggest that although exercise training is beneficial, diet remains an important modulator of glucose regulation, even in trained individuals, particularly when the exercise bout is not repeated. Rogers et al. (12) demonstrated using master athletes that repeating the exercise bout in a planned fashion was necessary for exercise to maintain an optimal glucose tolerance. However, here, the authors showed that restricting food intake in sedentary HFD-fed animals to produce comparable weight gains to trained HFD-fed mice elicited a similar blunting of glucose intolerance to that found in the trained mice, suggesting that a dietary stressor is a critical consideration.

Nonetheless, the concept of a protective “lag” effect following exercise training remains intriguing. Beyond confirming exercise as the primary causal factor, future investigations should aim to determine how long the protective effects of prior exercise training remain upon the cessation of training and consumption of a HFD, the minimum training duration required to replicate the effect and whether the duration and extent of protection responds to longer training periods and alternative exercise modalities. Of greatest interest though is the need for a physiological mechanism to explain the findings of Snook et al. (13). To this end, the design of preventative therapies would be aided by elucidating the exercise-induced molecular adaptations required to evoke the protective “lag”. Moreover, while the authors examination of adipose tissue...
revealed little of significance to define the increase in ΔTEE following exercise training, the demonstration that trained mice exhibited greater combined plantaris, EDL, and soleus muscle mass highlights skeletal muscle as a candidate tissue requiring additional exploration. Skeletal muscle represents the major tissue for whole body substrate disposal (2, 14), capable of undergoing substantial morphological and molecular alterations in response to exercise training. Loss of skeletal muscle insulin sensitivity causes systemic perturbations in glucose homeostasis and precedes the development of Type 2 diabetes (9, 15). A focus on skeletal muscle may, therefore, aid efforts to advance our understanding of how prior exercise training protects the body from exposure to a short-term high-fat diet.

DISCLOSURES
No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS
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